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Monitoring cardiogenic shock: echodynamics vs. pulmonary artery catheter Viewpoints from two leading experts

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As cardiogenic shock (CS) gains renewed focus through the SCAI classification[1] and a precision medicine approach, haemodynamic monitoring is at the forefront of advanced cardiac care. In this interview, Dr Simone Frea (Città della Salute e della Scienza, Turin, Italy) and Ass. Prof. Dr Jacob C. Jentzer (Mayo Clinic, Rochester, USA) explore the evolving roles of echocardiography and pulmonary artery catheterization (PAC), sharing their insights on how these complementary modalities are redefining clinical decision-making in the management of CS.

In recent years, there has been increasing interest in CS. In your opinion, how has the clinical approach to haemodynamic monitoring evolved? And what tools do you consider essential for a personalized management?

J.C. Jentzer: interestingly, the tools themselves haven't evolved much in the past decade, but our understanding of their use has changed. We still rely on PAC, echocardiography, arterial pressure monitoring with pulse contour estimations of stroke volume, and central venous pressure and oxygen saturation. However, there is renewed interest in titrating care using objective data derived from these measurements. We have also realized that CS is highly heterogeneous: haemodynamic phenotypes vary, and treatments must be tailored according to haemodynamics. While many of these concepts are decades old, they were not previously integrated into routine management and were not applied to the day-to-day care of CS patients. We now routinely consider the congestion profile and vascular tone for phenotyping of CS patients and incorporate how these change in response to therapies to guide care in a sophisticated manner at the bedside integrated with markers of tissue and organ perfusion.

S. Frea: the use of PAC declined significantly following the ESCAPE trial,[2] which, although not focused on CS, influenced clinical practice broadly. Interestingly, patients in the PAC arm showed lower left ventricular filling pressures, likely due to more effective vasodilator therapy. More recently, data from registries and the CS Working Group have reaffirmed the need for complete haemodynamic profiling, which consistently outperforms incomplete assessment in guiding individualized therapy.[3] This has led to growing interest in echocardiographic parameters such as echo-derived pulmonary artery pulsatility index (PAPi) and cardiac power output, which, if validated, may support the use of echocardiography as an initial or even alternative tool to PAC in selected patients. All CS patients need an arterial line, central venous access, and a high-quality echocardiographic exam—performed immediately and repeated frequently. However, while PAC provides robust macro-haemodynamic information, it offers limited insight into end-organ perfusion and congestion, where echocardiography excels by enabling dynamic assessment of renal, pulmonary, and splanchnic circulation. This approach requires continuous access to skilled operators, and in their absence, PAC remains essential. Ultimately, reliable haemodynamic data are not optional—they are a compass to sail in the sea: we need to know where we are going and to measure the response to treatment.

Dr Jentzer, although the integration of invasive haemodynamic monitoring and echocardiography provides a complete clinical picture in CS, which type of monitoring do you prioritize?

J.C. Jentzer: there is no single answer, at least in my practice. Generally, when managing patients with CS requiring significant vasopressors, inotropes, or mechanical circulatory support (MCS), I prefer

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the minute-to-minute haemodynamic evaluation a PAC provides, particularly when I have access to a continuous cardiac output/oximetry catheter. However, before they reach that point, echocardiography is useful both for initial diagnosis/screening and reassessment as clinical status changes. Of course, the data from echo and PAC are complementary. We often question whether echo measurements are truly accurate and precise, which is why the PAC remains the gold standard assuming that measurements can be done accurately and reproducibly. But echocardiography gives broader data, including 2D assessments of chamber size, geometry, and function, which are invaluable even without detailed Doppler haemodynamics. Doppler provides beat-by-beat analysis, showing cardiac performance at a single-beat level and illustrating how the heart performs under stress. While compensatory mechanisms such as tachycardia can maintain normal cardiac output even with low stroke volume, echocardiography can detect this change, showing how poorly the heart is truly functioning. Therefore, I see PAC and echo as complementary but used during different phases of the illness. My first move is usually echocardiography—it's fast and portable. Once in the ICU, especially in less severely ill patients, central venous pressure and central venous oxygen saturation may be sufficient initially to guide haemodynamic therapy. If the patient is sicker or not responding as expected to therapy, I escalate quickly to PAC to allow complete haemodynamic profiling. There are several specific conditions, such as predominant right ventricular failure and/or decompensated pulmonary hypertension, where early PAC use is my standard approach.

Dr Frea, in patients with deteriorating CS, some echo parameters might be less reliable and conditions rapidly evolve: when do you think invasive monitoring offers an advantage over echocardiography alone?

S. Frea: in such conditions, clearly the echo estimation of certain parameters, such as wedge pressure (WP), remains challenging[4]: the *E*/*e′* ratio performs poorly in the acute setting, as confirmed by disappointing results across multiple studies. In contrast, traditional parameters such as *E*/*A* ratio and deceleration time, validated in acute myocardial infarction, offer more reliable estimates. A multi-parametric approach combining cardiac indexes (e.g. deceleration time and tricuspid gradient) with extra-cardiac signs (e.g. B-lines on lung ultrasound) is probably the best strategy for estimating WP. In most patients, an echo-first strategy is feasible and effective, and PAC insertion should be reserved, for instance, for cases of atrial fibrillation or aortic regurgitation, which reduce reliability of echodynamics and may require invasive assessment. Similarly, poor response to first-line therapy or uncertain pulmonary pressures necessitate PAC. Moreover, the more advanced the SCAI stage, the greater the need for PAC use, particularly before, during, and after MCS. During weaning, however, I think the best approach is to use both echo and PAC. Crucially, PAC data must be collected and interpreted correctly: numbers alone do not guide care unless they are integrated into a treatment strategy. In early-stage CS, especially in acute decompensated heart failure (ADHF) patients, that generally have a slower trajectory, echocardiography is sufficient. In contrast, worsening acute syndromes, fulminant myocarditis, or acute myocardial infarction–related CS typically require early invasive monitoring.

Dr Jentzer, the PACCS trial is comparing early with no or delayed PAC use in ADHF-CS. Could you give us some insights on this trial?

J.C. Jentzer: although I'm not actually part of the PACCS trial, I personally see it as a strategy trial more than an intervention trial. Unlike studies that randomize patients to receive or not receive a therapy, PACCS examines the timing of PAC placement during the clinical course of CS. The hope is that early PAC use by experienced, multi-disciplinary teams will facilitate faster haemodynamic stabilization and potentially improve outcomes compared with reactive use in deteriorating patients. Observational data have suggested such benefits, although this remains unproven in randomized trials. However, I am sceptical about the use of any monitoring or diagnostic tool not directly linked to a potentially beneficial therapy, with the counterexample being coronary angiography, which can lead to PCI if needed. Early PAC placement may lead to earlier initiation of MCS, but the evidence for MCS in heart failure-related shock is not as robust as for infarct-related shock.[5–7] For ECMO and Impella, no randomized data are available in this setting, and for the intra-aortic balloon pump (IABP) we have the recently published Altshock-2 trial, which was underpowered, but showed no significant benefit.[8] If early PAC use simply increases the use of ineffective therapies, it may not improve outcomes. That is part of the challenge that we saw in the ESCAPE trial, where PAC use sometimes led to unnecessary or harmful interventions.[1] That risk persists unless PAC data are interpreted with clinical expertise and used within a structured decision-making framework, which is missing in most trials. Given the high level of expertise at participating centres in the PACCS trial, I would expect that if a benefit of PAC can be demonstrated, they will have the best chance of doing so.

It could be argued that an important future trial in CS would compare a specific treatment algorithm, using whatever monitoring data are necessary, with routine care. This approach has already been examined, for example, with diuretics.[9] The real opportunity lies in trials that use PAC to guide care toward well-defined haemodynamic goals. I recently addressed the concept of achieving normal haemodynamics and whether using PAC will compel us to normalize haemodynamics in patients who may not need it, for example, those with ADHF.[10] Altshock-2 proposed structured haemodynamic goals to define compensation—this approach may offer a better framework. Rather than rigid algorithms, establishing goal-oriented benchmarks—e.g. normal lactate, adequate urine output, and acceptable blood pressure—could guide therapy. If early PAC use enables more rapid achievement of these goals, improved outcomes might follow. Without that, demonstrating a benefit of early PAC becomes difficult. We must recognize that, while PAC provides data, it is only useful when tied to meaningful, goal-directed interventions.

Dr Frea, concerning mixed shock presentation (e.g. overlapping cardiogenic and vasoplegic components), how can echocardiography help identifying them, and to what extent can it guide subsequent therapeutic adjustments?

S. Frea: diagnosing mixed shock requires both systemic inflammatory response syndrome (SIRS) criteria and distributive haemodynamic

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features. A key early sign of transition from cardiogenic to mixed shock is a rising aortic velocity-time integral (VTI) accompanied by increasing lactate levels—even when blood pressure remains unchanged.[11] This haemodynamic pattern often precedes laboratory signs of inflammation by several hours. Calculating systemic vascular resistance from echo-derived stroke volume adds diagnostic precision. In our centre, we frequently observe this profile, which is accompanied by the need for switching vasodilators to norepinephrine, with infection markers such as C-reactive protein and procalcitonin appearing 6–12 h later. Thus, by monitoring lactates, pseudo-normalization of central venous oxygen saturation, and paradoxical increase in VTI, one can precociously detect the evolution to a mixed shock at an early stage. Cardiogenic shock remains a clinical diagnosis, but echocardiography is essential for aetiological clarification and phenotyping. For instance, in a SCAI stage B patient with a dilated left ventricle and severe functional mitral regurgitation, vasodilator therapy alone can improve cardiac output—easily assessed by echo if VTI and stroke volume rise by 20% without inotropes. Velocity-time integral is therefore central to both diagnosis and monitoring, with strong associations to in-hospital mortality, as shown by Jentzer *et al.*.[12] A shift from lactate-guided therapy to VTI-guided monitoring may be warranted, given the faster response time. I find this parameter particularly useful in Impella-supported patients: an increase in native stroke volume during device weaning—provided that WP remains stable—is a sign of left ventricular reserve. This example illustrates an integrated strategy in which echocardiographic stroke volume, combined with PAC-derived filling pressures, enables individualized and safe MCS withdrawal.

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